

neutralized the $TNF-\alpha$ protein. The reaction of these mice to ozone was similar to that of the resistant mice. Although Kleeberger calls this evidence “intriguing,” it is not proof that $TNF-\alpha$ is the controlling gene in ozone susceptibility, he says. More linkage and physical map studies are needed to identify what he suspects may be a bevy of major and contributing genes associated with differing susceptibilities to ozone.

Indeed, Aravinda Chakravarti, a professor of genetics and medicine at Case Western Reserve University in Cleveland, Ohio, questions the use of mice as surrogates for how ozone affects human biology. The inbred mice used in the studies surely have less genetic variation in their reaction to ozone than that found in humans, he says, and moreover, humans metabolize oxygen differently.

But Kleeberger points out that the “mouse represents a unique model to study genes in their most simple representation.” Michael Blaese, chief of the clinical gene therapy branch at the National Human Genome Research Institute in Bethesda, Maryland, says it makes sense that $TNF-\alpha$ may be in some way linked to ozone susceptibility because it is involved in so many important biological activities.

Even so, Blaese believes that any effort to clinically correct or repair susceptibility genes will be a long time in coming because of the inherent difficulties of gene therapy. More likely, he says, researchers will search for clinical ways to interfere with the protein encoded by the gene.

This work offers hope to millions of asthmatics, says Sharon Hipkins, director of programs and policy at the Asthma and Allergy Foundation of America in Washington, DC. It highlights “clinical recognition that there probably is a familial trait that mediates reaction to high ozone levels and that, likely, many asthmatics are affected,” she says. But Kleeberger says that asthma is a very complex disorder with a number of different phenotypes and that the association between ozone and asthma susceptibility is not completely understood.

“What is most interesting about these studies is that we have always viewed air pollution as having adverse effects on society as a whole,” says Drazen. “Like reactions to medications, we are learning we might be able to identify selected individuals who are affected.”

EHPnet

Mad Cow Media

“Mad cow disease,” the common name for bovine spongiform encephalopathy, or BSE, is alone enough to generate concern in anyone who consumes the daily news along with his hamburger. When the disease broke out among humans in 1996, there was panic among the public, who feared that eating beef would result in death from the “brain-rotting” disease. To help combat such public hysteria, the private charitable Sperling Foundation established the Official Mad Cow Disease Home Page, located at <http://www.mad-cow.org/>. The page is a repository for over 3,650 articles on BSE and related pathologies, and is updated twice a week (hourly during breaking events). News clips from a vast array of sources are warehoused and cataloged according to topic.

The Official Mad Cow Disease Home Page is managed by Tom Pringle, scientific director of the Sperling Foundation’s Creutzfeldt-Jakob disease (CJD) program



(CJD is commonly known as the human equivalent of BSE). Says Pringle of the site, “Basically, my ambitions for the site were to create a new paradigm for human disease research, based on unique aspects of the Web such as unlimited available space and essentially no distribution costs. This allows much more depth in terms of articles archived, much more frequent updating than a fixed journal, room for supplemental commentary and clarification by appended correspondence with scientific authors, and opportunity for value-added background links.”

The home page features an extensive list of links to articles divided by topic into 10 categories: General News; Prion Molecular Biology; Victims; Prion Research; BSE in Blood, Milk, Meat; How Beef is Made; Alzheimer’s and CJD; Other Cow-to-Human Diseases; Epidemic; and Mad-Cow Scientists. These articles, culled from the lay press, present a chronicle of the development of the mad cow disease scare and research that is being done to identify the source of this alarming malady. The home page also lists links to several different information resources, along with a site search engine and technical e-mail correspondence and sharing of news with Pringle.

Over 200 links to different online journals, search engines, databases, and other resources are listed from the home page under The Best Links. Some of these links are for general Internet reference areas, while others are specifically relevant to the topic of BSE, such as the Food Safety and Inspection Service’s listing of food recalls from 1990 through 1998. This page also contains several links to online molecular biology tools, such as the University of Illinois’s NCSA Biology Workbench.

The Real Science link on the home page leads to a list of links to scientific papers on the study of BSE. The papers are grouped on the page by topic, with each link leading to an abstract and citation for the original paper. This link also includes reviews by Pringle of current research and tables—updated regularly—of known mutant strains of BSE. Pringle posts his own research as well, saying, “I strongly believe that this is the future of biomedical research and an ethical mandate in the human disease context.”

The 3D Interactive Prions link allows visitors to view two- and three-dimensional renderings of the prion protein molecule. Finally, the home page includes a link to the Fundraising and Support for CJD Victims page, a list of support groups for CJD sufferers and their families.